A HISTOPATHOLOGICAL AND BACTERIOLOGICAL STUDY OF ATROPHIC RHINITIS

THESIS

FOR THE DEGREE OF
MASTER OF SURGERY

(OTORHINOLARYNGOLOGY)

BUNDELKHAND UNIVERSITY
JHANSI (U.P.)

2000

SUBHASH CHANDRA

DEDICATED
TO
THE LOVING MEMORY
OF
MY FATHER

CERTIFICATE

This is to certify that this thesis entitled "*A HISTOPATHOLOGICAL AND BACTERIOLOGICAL STUDY OF ATROPHIC RHINITIS*" is a bonafied work of **Dr. SUBHASH CHANDRA** conducted in the department of ENT, M.L.B. Medical College Jhansi.

He has put in the necessary stay in the department as required by the regulations of the Bundelkhand University Jhansi (U.P.).

Dated : 29/2/2000

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SUBHASH CHANDRA

CONTENTS

INTRODUCTION

INTRODUCTION

Atrophic Rhinitis is a chronic inflammatory condition of nose characterised by atrophic changes of mucosa of nose, and underlying bone of the turbinates and very roomy capacious nasal fossae, the formation of thick crusts and foetor to which is attributed the term ozaena. The main presenting feature of the Patients includes, Dryness of nose, Crust formation, Nasal obstruction, Headache, Nasal discharge, Epistaxis, Anosmia.

The aetiology of atrophic rhinitis is still unknown. Infection, endocrine imbalance; deficiency of iron, zinc and fat soluble vitamins, wide breath of nasal cavity and small antra; and at times DNS are contribute to atrophic changes in nasal mucosa while chronic sinusitis, lupus, tuberculosis, syphilis, leprosy, surgery and accidents have role in causing atrophic rhinitis.

Fouad et al (1980) studied cellular immunity in the patients with atrophic rhinitis. There was altered cellular reactivity or loss of tolerence to nasal tissues.

The present study has been under taken a view of the above mentioned facts with the following objectives:

- Prevalence of primary atrophic rhinitis and secondary atrophic rhinitis.
- Prevalence of different Baterias in atrophic rhinitis.
- Histopathology of nasal mucosa in atrophic rhinitis whether it is purely bacterial infection or associated with auto immune disease or other diseases.
- Radiological examination of paranasal sinuses in correlation of atrophic rhinitis.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

A century is passing by since FRANKAEL in 1876 described this chronic distressing condition, incurable yet not fatal. Inspite of tireless efforts by many, the code of its aetiology still remains undeciphered. Many theories and hypothesis are put forth to explain this condition but have failed to catch general acceptance.

Anatomical Considerations

The area under consideration "Nasal Cavity and Paranasal Sinuses" has a complex configuration. Anatomy of paranasal sinuses has been known for several centuries.

Rigmure (1651) defined the maxillary and frontal sinuses. Gross anatomical relation's of the paranasal sinuses are shown in figs. A, B, C and D (Friedman and seborn, 1982).

Normal Histology

The anterior part of vestibule is lined by Keratinizing squamous epithelium which is a continuation of the skin covering the external nose. The lining on being traced backward becomes pseudostratified, ciliated columnar type which is characterstic of respiratory epithelium. This lining is called Schneiderian membrane after the name of the histologist who was the first described it in 1660. Interspersed amongst the ciliated cells are varying number of goblet cells which are identified as unstained areas under the light microscope.

FIG. A

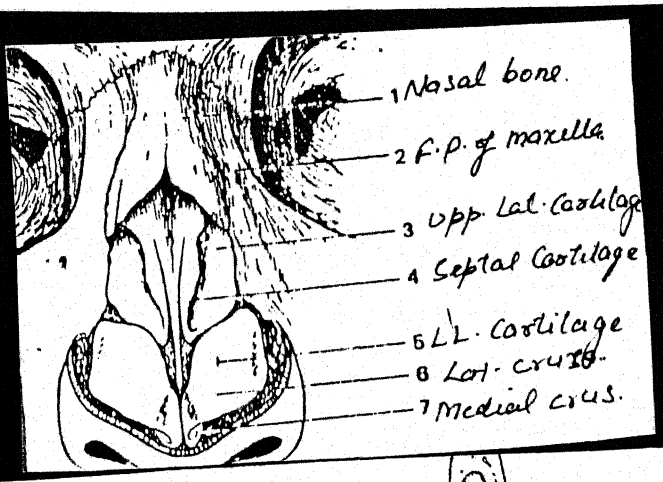


FIG. B

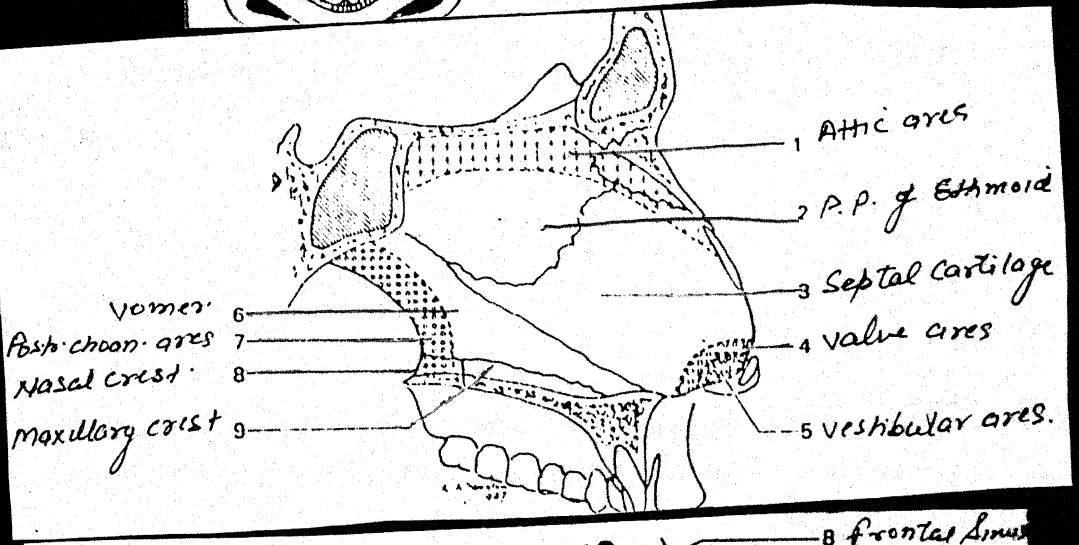


FIG. C

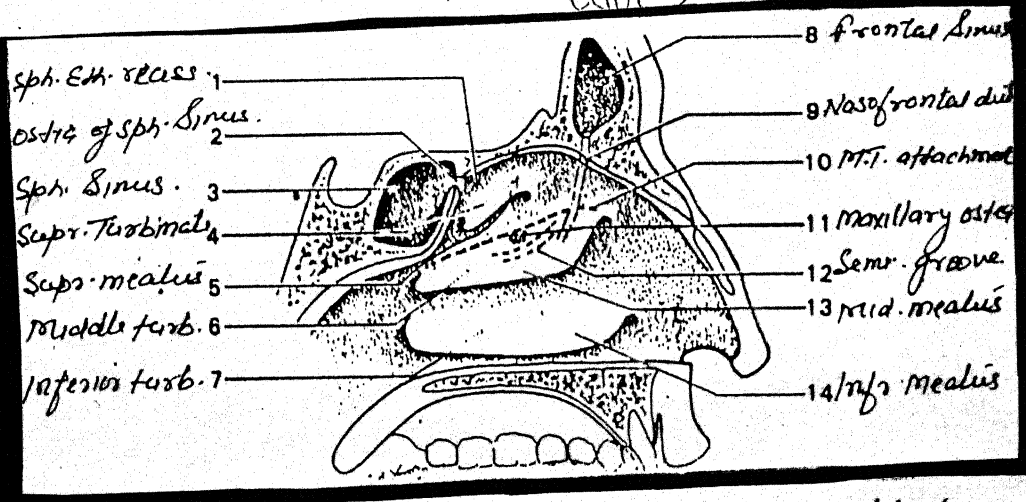
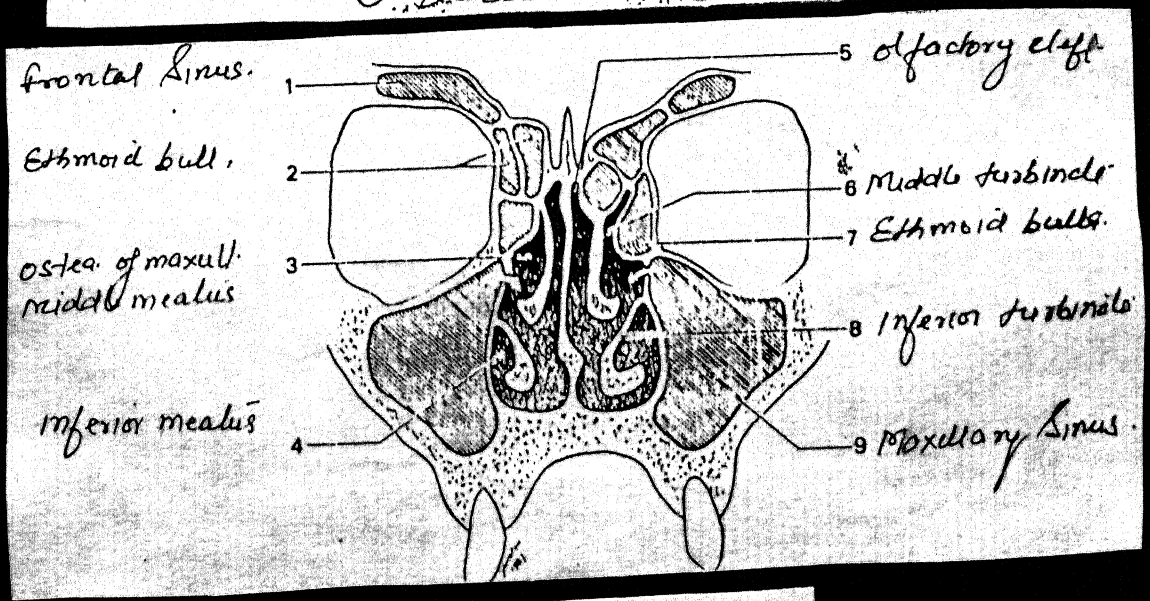


FIG. D



ANATOMY OF NOSE

Numerous mucosal gland of mixed nature are found deep in the lamina propria. The ductular system is not well developed and some times may be difficult to identify on the periphery of these glands are located the myoepithelial cells. The importance of these cells is in the pathogenesis of certain mucosal gland tumours.

Paranasal mucosa is similar to that of the nasal fossa, though there are slight differences, the columnar cells tend to be shorter resulting in thinner epithelium. These cells bear cilia and there are large number of microvilli. The lamina propria also tends to be thinner and usually bears fewer mucosal glands (friedmann and Seborn, 1976).

NASAL ERECTILE TISSUE

An interesting feature of the lamina propria of the nasal and occasionally paranasal sinuses mucosa is the presence of complex blood vessels which are designated as "ercetile tissue". The vessels contain substantial amount of smooth muscle fibres which are arranged in spiral fashion, thus giving rise to irregular arrangement an cross section. These muscle fibres are under the influence of the autonomic nervous system and also react to chemical reagents, including some hormones. Another interesting aspect of the nasal cavity is the existance of melanocyte's which have been demonstrated in the skin of the vestibule (Szabo, 1959) and the epithelial lining of the nasal cavity (AZK and Lawson, 1974).

OLFACTORY MUCOSA

The human olfactory area extends from the roof of the nasal cavity about one centimeter down wards on either side of the nasal septum and on the medial surface of superior turbinate. This area is covered by pseudo stratified epithelium. The olfactory cells are essential bipolar receptor neurones and are provided with distal and proximal processes. The distal process pass through supporting cells towards the surface and are called dendrite. The proximal process is essentially an axon. They form synapse connections with neurone of second order in the olfactory bulb (Lenz, 1977).

INCIDENCE

Atrophic Rhinitis is included as one of the disappearing diseases in the Western countries. Western authors attribute this change to their improvement in living standards. In our country, the disease is still prevalent and Jain (1966) reported a frequency of one case of atrophic rhinitis for an average of 1700 new out patients in Kakinada. He is of the view that the climate, poor hygienic condition and persistance of infections diseases like syphilis and leprosy are all major contributing factors in keeping the disease alive in this country. The disease is more prevalent in yellow races, occurs in Negroes of South, Central and North America, but is rare in Negroes of Africa Oceania and West Indies (St. Clair Thompson).

In 1917, Roy (quoted by Shapiro 1967) published the results of his observation on the incidence of atrophic rhinitis among the various races. He examined the nasal cavities of 5000 negroes in Africa and did not find a single case of atrophic rhinitis. On the other hand Negroes and other people of mixed blood in Brazil as well as in other countries of South and North America Eskimoes, Malayes, Filliphines and North American Indians showed a considerable incidence of this condition.

Girgis (1966) recorded an incidence of 117 cases of atrophic rhinitis in 11365 out patients in United Arab Republic. He observed that the disease was more common in poor citizens.

AETIOLOGY

Atrophic Rhinitis is regarded as a disease of the young subjects, peak of incidence between the ages of 10-12 years. The disease shows a prediliction to the females than males. Females are affected 5 times more frequently than males (James, 1963).

Atrophic Rhinitis has been divided into two groups on the basis of the causative factors (James – 1965)

(I) Primary atrophic rhinitis

(II) Secondary atrophic rhinitis

Primary atrophic rhinitis includes those cases, where the exact cause is unknown. It is quite possible to consider multiple factors to produce this condition.

Secondary atrophic rhinitis consists of the condition resulting from syphilis, Tuberculosis, lupus vulgaris, leprosy and scleroma or operative destruction of the nasal mucous membrane.

The various views about the probable causes of Primary atrophic rhinitis may be discussed on the following headings : (Girgis 1966, Shapiro 1967)

(i) ***Sinus Infections*** :

Grunwald, Lautenschlager, harry L. Pollack were of the opinion that Atrophic rhinitis is always secondary to sinus infection. In chronic sinus infection, the secretions act as an irritant to the connective tissue which proliferates, later resulting in cicatrization, fibrosis and atrophy (Girgis 1967, Ballenger (1969).

James (1965) attributed the cause to a proceedings severe nasal inflammation with associated vitamin nutritional and endocrinal deficiency. Nasal Diptheria and suppurative rhinitis and sinusits of Measles and other exanthemata are considered to be the common anticidents. Those severe infections have been shown to cause necrosis of the glandular and ciliated epithelium and subsequent repair by fibrosis and metaplasia.

(ii) ***Hypertrophic Rhinitis***

Bosworth regarded Ozaena as secondary to hypertrophic rhinitis, Lautenschlager, Eggston and wolff considered that all cases of Atrophic rhinitis were due to chronic infection, which induced periarteritis and endarteritis of the nasal mucosa producing atrophic changes.

(iii) ***Bacterial Infection***

This remains probably the oldest theory in literature. Many organisms have been isolated and accused of responsible for this condition. Pseudo-Diphtheria bacillus (Belforiti and Dellavedova), coccobacillus foetidis Ozaena (Perez), Bacillus mucosus (Abel) Bacillus foetidis Ozaena (Hazek) and other organisms. The infectivity and pathogenecity of these organism could not be proved. They are possibly secondary invaders and may be responsible for the foetor.

(iv) ***Developmental Theory***

Excessive patency of the nasal cavities in relationship to the skull has been put forward by Hopmann, Siebenmann, Garbar and J. Wright. Peste (1949) demonstrated that the large nasal fossa in atrophic rhinitis are at the expense of small under developed antra. The author concluded that the poor pneumatisation of antra is probably the decisive factor in the pathogenesis of the disease.

Stanur showd that the length of nose measured between the anterior and posterior nasal spine is shorter in people suffering from ozaena than in normal persons. He attributes it to the stand still of the development of facial skeleton.

Wachsberger (1934) stressed the importance of abnormal width of the nasal cavities, which increased the evaporation of the already viscus sceretions of the ozaena resulting in crust formation.

Shapiro (1967) believes that the abnormal width of the nasal fossa is more likely to be in the nature of an effect than the cause. Supporting this is the fact that the condition is very rare in African Negroes, who have wide short nostrils and much common in Asiatic who have relatively narrow nostrils.

(v) ***Hereditary Theory***

Fleishmann believed that it is due to hereditary inhibition of the nasal mucosa, which can run according to Mandelian law. atrophic rhinitis among the same member's of the family is reported in the literature.

(vi) ***Endocrine Theory***

The occurance of the disease mainly during puberty, is predilection to the female sex and its worsening during menstruation and pregancy suggest its relationship to endocrine factors (Lautenschlager 1924, Pratt 1931) Mortimer and his associates by experimental work

postulated a decreased secretion of gonadotrophic hormones at puberty and also a lack of response of mucous membrane to normal oestrogen circulating in the blood. Many recent authors like James (1965) and Ballenger (1969) do not consider this factor in the aetiology of atrophic rhinitis. The alleged role of endocrine dysfunction has fallen into disrepute from lack of evidence (Bernet, 1965).

(vii) ***Deficiency Theory***

The disease is more prevalent among the poor class of people and refugee camps. The deficiency of mainly attributed to iron and fat soluble vitamins especially vitamin A (Girgis 1966).

Bernat 91968) observed that iron therapy was successful in atrophic rhinitis, if the nasal mucosa is not irrepairably damaged. He concludes that iron deficiency has a definite role to play and improving the diet can reduce the incidence. Gadre et al (1971) observed mild anaemia in their cases. However, Barkve (1968) was unable to find any incidence of iron deficiency in his studies.

Zinc deficiency may manifest as reduced smell and taste sensations (Condas et al, 1977, Boyette, 1982). It is indispensable to cellular function and division (Boyette, 1982) and is essential for the activity of serum alkaline phosphates. Because of this, low levels of alkaline phosphatase can be expected in association with hypozincimia

(Prasad et al, 1978 & 1979, Roth & Kirschgnesner, 1974) and therefore alkaline phosphatase activity may be used an index of clinical zinc deficiency and a monitor of therapy (Kaserkis & Schuna, 1980).

Hollender (1944) believe that trauma, during effect of inspired air and reduction in the blood supply are the principle factors. Turner (1968) also supports the theory that reduction in the blood supply are the principle factors. Turner (1968) supports the theory that recduction in blood supply to the nasal mucosa is the determining factors in atrophic rhinitis.

Jackabfi (1954) considers autonomic dysfunction to be of primary importance for producing atrophic changes, creating suitable conditions for the colonisation of the mucosa by specific capsulated bacteria which than become pathogenic.

All said it seems that the disease is due to more than one factor most probably due to some hereditary or endocrine factors and that ozaena is caused by secondary invasion by saprophytic organism (Girgis 1966).

PATHOLOGY

Atrophic conditions seems to be generalised than localised to the nasal mucosa. Pharynx, larynx, Trachea and Bronchi are involved

frequently and atrophic vaginitis is also reported. Girgis (1966) considers atrophic rhinitis related to the syndromes like plummer vinson and scleroderma, fundamentally characterised by dystrophic conditions of mucosal tissue of ectodermal and mesodermal origin.

A good account of the pathology is given by Eggston and Wolf (1947), Taylor and Young (1961).

In the early cases the histopathological picture is that of chronic non specific inflammation. The pathological changes are found in all the elements of the nasal mucosa, namely the epithelium, basement membrane, Tunica propria and even in the underlying bone. The columnar ciliated epithelium undergoes metaplasia to squamous epithelium. All stages can be founds the cells loose their cilia, begin to flatten, goblet cells become fewer and finally a mass of keratin may be seen over the surface.

While Taylor and young (1961) have found the basement membrane when present to be thin, others like Shambaugh (1931) Hollender (1944) have noted thickening of this membrane with more collagen. It is due to an inflammatory process which produces endarteritis and periarteritis of terminal arterioles (Ruskin, 1942, Taylor and Young 1961 and Holopainen, 1967). Many authors have stressed the presence of endarteritis obliterans but German investigators deny this.

Characterstics changes are the dilatation of capillaries. The endothelial cells have more cytoplasm and shows a more positive reaction for alkaline phosphate (Taylor and Young 1961). High concentration of Alkaline phosphatase explains the absorption of bony nasal turbinates.

Sinus show definite diminution of size and this is due to arrest of development as the disease starts in young age. The mucosa of sinus shares in the atrophic changes but no crusting occurs. Bony wall of maxillary antrum shows evidence of sclerosis (Girgis 1966).

CLINICAL MANIFESTATION

Clinical features of atrophic rhinitis are typical and are well described by James (1965) Logan Turner (1965) , Ballenger (1969).

The most characterstic feature is the cadaveric smell or stench from which the disease derives its name, Ozaena.

In women the foetor becomes more during menstruation and pregnancy. Anosmia of varying degrees are present, while Epistaxis, headache and sense of dryness of mouth are other common complaints. Nasal obstruction is due to accumulation of crusts but even without this the symptom may be complained due to inhibition of sense of passage of air (Girgis 1966).

Externally the nose of person having atrophic rhinitis shows presence of thickened vestibular rim and button like tip (Prond 1947). Depression of nasal bridge due to arrest of development and oblique furrows on either side of nose at the junction of bone and cartilagenous wall due to shrinkage and underdevelopment of the cartilage are usually found (Girgis 1966). Patient may be in drawn and shows evidence of Psychological upset.

Nasal cavities in early stages appear roomy, the mucosa pale and covered by viscid, greenish secretion. Atrophic changes begin's in the inferior turbinate and middle turbinate. Later the whole lateral wall becomes flattened by atrophy. Nasal cavities get wider and viscid secretion dry up producing greenish or black crust on the mucosa.

Pharynx usually shows atrophic changes and its posterior wall appear dry and glazed. Hoarseness of voice shows laryngageal involvement and indirect laryngoscopy reveals crusting in the inter-arytenoid and subglottic region. In the trachea and bronchi also scales may accumulate and in some cases obstruction persists even after tracheostomy and proves fatal (Girgis 1966).

In atrophic rhinitis the nose is affected Bilaterally but in the presence of gross septal deviation the condition may be absent in the narrow fossa. (Thomas and Negus 1955).

S.C. Gupta considered that atrophic changes are seen on the concave side and if deformity is gross, obstructive symptoms occurs on the convex

side of the septum. An excessively large air space in the nasal cavity is believed to be are the causative factors in atrophic rhinitis (1985).

TREATMENT

The various methods of treatment fall into two groups : Medical and Surgical and it can be confidently said the latter has completely eclipsed the former because of the comparatively lasting and definite result.

Medical treatment presents never ending list and are mainly in the form of proper nasal hygiene, alkaline nasal douches, glucose glycerine paste and oily drops.

Oestrogenic substance was tried based as theory of oestrogen deficiency as a cause of the disease (Blaisdell 1938), Streptomycin was used both systemically and as solution locally by Moselella (1950) with disappearance of crusting and smell. Sen used Nicotinic acid, the peripheral vasodilator with encouraging results with the same idea of nasal vasodilatation. Arnulf did some stellate ganglion block with good but temporary results. This was supported by the work of Sharma and Sardana (1965).

At present the attention is mainly concentrated on surgical treatments and constant efforts are provided to come out with a perfect procedure. The basis of the surgery has sprung from the observation of Sanger and

Sounderman (1894) that most nasal cavities resulted from using meatal obturator for the nostrils.

Most of surgical methods are aimed to reducing the nasal air way. The drying effect of inspired air is a major factor in causation or progression of the disease. Partial oblitration of nasal cavity breaks this viscious circle, set into action by the factors, which cause atrophy of the mucosa and thus widening of the cavity (Girgis 1966).

Review of the methods may be conveniently divided into

(i) ***Traumatic Procedures***

Lautenschlager (1917) was the first to describe the surgical technique to narrow the nasal cavity. By a caldwell Luc's approach, he reached the nasoantral wall, separated it upto the posterior part by chisel and hammer and pushed the whole segment medially against the septum.

Lautenschlager's procedure was further modified by Halle (1918) Wittmack (1919), Hensburg (1921) Wachsberger (1934) and Rethi (1948).

While Halle tried a simpler intra nasal approach for the mobilisation of lateral wall of the nose, Hens berg tried to keep the mobilised wall in the midline by suturing it to the septum with magnisium wires. But in most of

these cases the infractured segment tended to spring back to the original position.

Wittmack (1919) followed the lautenschlager technique and as an addition transplanted the stenson's duct in side the maxillary antrum with the idea of moistening the nasal mucosa by the saliva. Though there was clinical improvement, some patients were left with a distressing salivary fistulae.

(ii) ***Submucus Injections***

Begining of this line of treatment was marked by Gersung (1900) who injected liquid paraffin submucosally into the nose.

Paraffin injection were also done by Banstein but sloughing was a common feature.

Chung et al (1964) introduced medical silastic S-5392 which when catalysed and injected submucosally became solid in a few minutes. The material is clear some what viscid fluid, which when mixed with catalyst became solid of rubbery consistency in several minutes. The time required for this depends on the temperature, humidity and the amount of catalyst used.

(iii) ***Implantation Techniques***

Good range of work has been done on this line using various autogenous, homogenous and heterogenous materials from the beginning of this century. Harry L Pollock (1927) after having had disappointing results with Lautenschlager and Halle operations began displacement of septal flap towards the lateral wall of the nose with implantation of various autogenous material like fascia lata abdominal fascia, bone and cartilages.

Kelmer (1931) and Rasnetz (1939) have tried Ivory implants with instantaneous relief but extrusion of implants was common.

In Cairo, Iskander H. Girgis (1966) has done laborious work on this problem. He first started using autogenous living tissue flaps as material for implantation. Through a sublabial incision a local periosteo-fascial flap is raised from the cheek with a pedicle kept intact medially beside the 'Pyriform aperture of the nose. This is then tucked under the floor of nose. But he dropped this technique in favour of using Dermofat taken from anterior aspect of thigh, for narrowing the wide nasal fossa.

The latest to the addition of various surgical procedures for treating atrophic rhinitis is complete nostril closure advocated by Austin Young (1967) from Shefield. The skin of the vestibule is raised as a flap by an incision on the mucocutaneous junction and dissecting it forwards. This is than sutured in mid line. After a period of six months a complete closure on apparently normal mucous membrane is restored in the nasal cavity.

J.F. Neil (1967) followed the Young's operation in one case of unilateral atrophic rhinitis with deviated septum : Results is reported good.

Even after taking with various forms of treatment one really wishes to have a better solution for this chronic distressing disease.

MATERIAL AND METHODS

MATERIAL AND METHODS

This study was carried out in the department of Ear, Nose and Throat, M.L.B. Medical College, Hospital Jhansi from Jan., 1998 to Jan., 2000. The cases of atrophic rhinitis including in this study were only those who belonged to Bundelkhand region i.e. Jhansi, Jalaun, Hamirpur, Mahoba, Banda, Lalitpur, Datia, Tikamgarh, Shivpuri, Sagar, Beena, Chattarpur districts etc.

During this period a total number of 25 patients attending ENT department were included in this study. These patients were admitted in ENT wards and subjected to detailed history and examination. The patients belonged to both male and female sexes and were of different age groups.

All the patients were subjected to the clinical work up according to the following protocol.

Clinical Examination

Name :	Age/Sex
Religion	MRD/OPD No.
Occupation	Ward/bed
Date of Admission	Date of Discharge

A. Clinical Data

a. History

1. Complaints with duration

Weakness	Bleeding
Crusting	Dryness of pharynx & larynx
Ozaena	Maggots infestation of nose
Headache	Anosmia/Hyposmia
Thirsty feeling	

b. Past History

Trauma	H/o operation
Allergy	T.B./Syphilis
Leprosy	

c. Personal History

Use of snuff	Smoker
Socioeconomic status	Sinusitis

d. Family History

Drug treatment and operation

Loss of Sense of smell.

B. Physical Examination

1. General Examination

General condition

Pulse	Blood Pressure
Temperature	Paller
Oedema	Lymphadenopathy

2. Systemic Examination

- Central Nervous System
- Cardiovascular System
- Respiratory System
- Abdomen
- Evidence of distant metastases

3. Local Examination

General

Face

Upper 1/3	Middle 1/3	Lower 1/3
Loss of eyebrow	Nasal bridge deformity	

Ala – Nodular & Rubbery

Bridge < Normal / Swellen / Sunken

Anterior Rhinoscopy

- Vestibule - Loss of vibrisae
- Septum Perforation - Collumella
- Pyriform apperture - Any obvious abnormality

Leteral wall

(a) Inferior turbinate

(i) Atrophy

(ii) Anterior End

(iii) Mucous Membrane :

Colour,

Atrophy

Oedema

Discharge

Area of bleeding

(b) Middle Meatus

Middle turbinate

Discharge

Type

Amount

Site of collection of pus

Site of origin of pus

Mucous membrane :

Colour

Atrophy,

Oedema

Discharge

Polyposis

Posterior Rhinoscopy

(i) Post end of septum

(ii) Opening of Choana

(iii) Post free end of turbinates

(iv) Discharge

(v) Mass

(vi) Ethmoid recess

(vii) Roof of Nasophanyx

(viii) Adenoids

(ix) Eustachian tube opening

Transillumination test :

Maxillary antrum

Ethmoidal Sinus

Frontal Sinus

Examination of Throat

Regional lymphadenopathy

Any other findings

C. Investigations.:

Blood

Haemoglobin gm%, TLC Cells/mm

DLC : P % L % E % B%

ESR mm in one hour

Blood urea mg% Blood Sugar mg%

VDRL Test

Urine : Alb, Sugar

Nasal Pus - Culture & Sensitivity

Radiological examination

X-ray PNS – occipitomental view

X-ray skull Lateral view

D. Histopathological Examination

The biopsed material was fixed in formline. The processing was done with the help of histokinet.

Hydration :

The tissue were dehydrated by passing through different grades of alcohal as summarised below :

Formal Alcohol (90ml of 70^{0}C ethylalcohol + 10 ml of formaldehyde)	-	One hour
90% ethyl alcohol	-	One hour
95% ethyl alcohol	-	One hour
Absolute alcohol (2 changes)	-	One hour each
Copper alcohol (2 changes)	-	One hour each

Cleaning :

This was done by summing the tissues through aniline oil – 6 hours and xylene (2 changes) one hour each.

Impregnation :

This was achieved by passing the tissues through xylene + Paraffin wax for 1½ hours and paraffin wax for 1½ hours. After preparing the blocks with the help of L shape moulds of brass.

The section were cut at 4-5 mm thickness.

Routine haematoxylene and Eosine staining was done in each case according to technique described by Culling (1963).

The following protocol was followed while carried out the histopathological examination :

Histopathological Examination

Name

Age/Sex — Histopath Lab No.

CR No./OPD No. — Type of Specimen.

Gross appearance

Size — Colour

Weight — Shape

Consistancy — Cut Surface

Solid/Cystic — Any other special feature

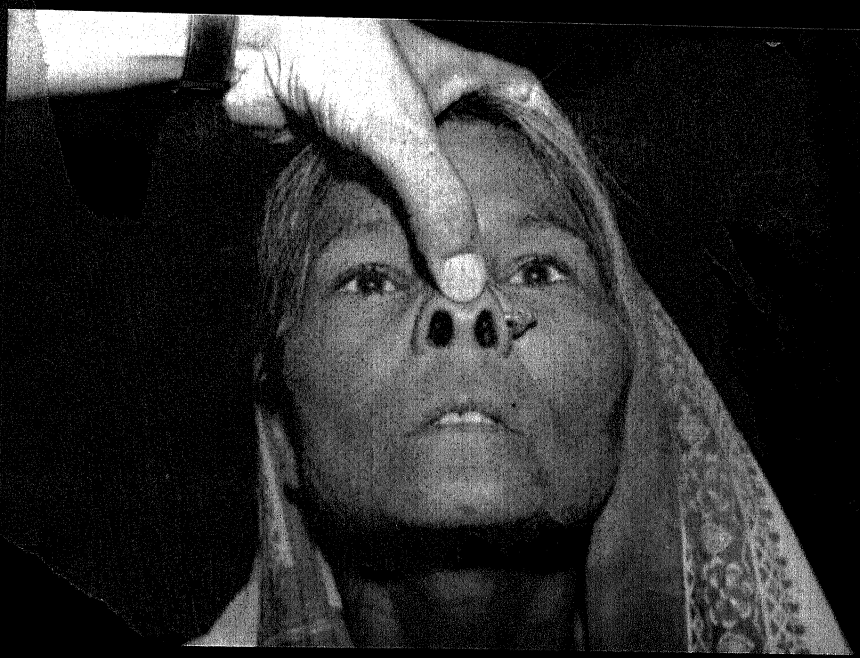
Microscopic :

Histopathological findings.

METHOD OF TAKING BIOPSY

INSTRUMENTS USED IN TAKING BIOPSY

BENIBAI - 53 YEARS FEMALE
A CASE OF ATROPHIC RHINITIS



SAME PATIENT SHOWING NOSTRILS

OBSERVATIONS AND RESULTS

OBSERVATIONS & RESULTS

The present study consisted of 25 cases of atrophic rhinitis who attended the Department of E.N.T. at M.L.B. Medical College Jhansi during the period of January 1998 to January 2000.

The following observations were made in their clinical presentation and follow up from 25 cases upto 24 months.

Incidence

During the above period in all 24,994 cases attended the ENT OPD and in 25 patients nasal biopsy specimens were taken for histopathological diagnosis and nasal swab was sent for bacteriological study. Nearly 100 cases of atrophic rhinitis attended the OPD and they refused for proper investigation. Therefore overall incidence of atrophic rhinitis was 0.5% in E.N.T. O.P.D.

Age Incidence

The youngest of the series was 11 years old and the oldest was 68 years. Age Incidence is shown in Table No.I

Table No. – I

Age group in years	Number of cases	Percentage
1-10	-	-
11-20	4	16%
21-30	6	24%
31-40	5	20%
41-50	3	12%
51-60	6	24%
61-70	1	4%
Total	**25**	**100%**

Sex Incidence

The number of patients belonging to either sex is shown in Table No.II.

Table No. – II

Sex	No. of Cases	Percentage
Male	7	28%
Female	18	72%
Total	**25**	**100%**

Table No.II shows the atrophic rhinitis was predominantly found in females. The female-male ratio was 18:7.

Occupational Incidence

In the present study majority of the patients were housewives. Other's were farmer's or students.

Table No.III shows the occupational incidences.

Table No. – III

Occupation	No. of cases	Percentage
Housewife	16	64%
Farmer	6	24%
Student	2	8%
Bidi maker	1	4%
Total	**25**	**100%**

Religion

Atrophic Rhinitis is more common in Hindus.

Table No. – IV

Religion	No. of cases	Percentage
Hindu	22	88%
Muslim	3	12%
Christians	-	-
Others	-	-
Total	**25**	**100%**

Atrophic Rhinitis is found 88% in Hindus and 12% in Muslims in this series. None patients of other Religion was seen in this study.

CLINICAL PRESENTATION

Symptoms

Table No.V shows the various symptoms complained of by the patients.

Table No. – V

Symptoms	No. of patients	Percentage
Crust in the nose	25	100%
Foetor	25	100%
Nasal blockage (obstruction)	23	92%
Epistaxis	10	40%
Anosmia	24	96%
Hyposmia	01	4%
Headache	19	76%
Myiasis of Nose	10	40%
Dryness of pharynx or throat	20	80%
Hoarsness of Voice	-	-
Frequent attack of cold/sneezing	7	28%
Unilateral	1	4%
Bilateral	24	96%

The presenting symptoms in all the cases were crusting, Foetor, dryness of pharynx or throat and Anosmia. Crusting was seen mostly when the patients blows out the nose while the foetor was perceived mostly by their relatives and neighbours. Headache, Epistaxis and Myiasis of nose were other frequent complaints.

Duration of Illness

The duration of illness in the patients under the study shown in the Table No.VI.

Table No. – VI

Duration of Symptoms in year	No. of Patients	Percentage
Less than 1	3	12%
1 – 5	8	32%
6 – 10	11	44%
More than 10	3	12%
Total	25	100%

Majority of the patients (56%) presented with disease since long duration and these patients had various types of medical treatment for long time without relief.

Table No.VII shows various signs noted in their patients.

Table No. – VII

Symptoms/Signs	No. of Patients	Percentage
Foeter	25	100%
Crusts in the Nose	25	100%
Dryness of nasal mucosa	25	100%
Nasal discharge	16	64%
Deformaities of the Nose	7	28%
Widened Nasal Cavity	24	96%
Maggots in nose	10	40%
Septal perforation	4	16%
Maggot in soft palate	1	4%
Maggot in tonsillar fossa (Rt)	1	4%
Atrophy of pharyngeal mucosa	20	80%
Anterior end of middle & Inf. turbinates atrophy	25	100%
Colour of nasal mucosa pale	24	96%
Deviated Nasal Septum	1	4%
Posterior end of turbinates – atrophy	25	100%
Posterier end of septum (thin) – Erosion	12	48%
Opening of Choana – wide	25	100%

Foetor, Crusts, dryness of the nasal mucosa, atrophy of turbinates, atrophy of pharyngeal mucosa, wide opening of Choana and widening of the nasal cavities were present near about in all cases. Crust varied in amount in different individual.

Perforation of the septum was present in 16% cases. But in all these cases it was due to erosion of septum by Maggots in the nose. Crusting of the posterior pharyngeal wall was present in none of the case while maggots in tonsillar fossa (Rt) was present only in one patient (4%). And in one case maggots were present in soft palate.

Hereditary History

We had studied thirteen (52%). Their family members were suffering from the same disease. Rest of the cases failed to show any family background for the disease (48%).

Illness associated with this condition

Out of the 25 cases, 10 cases had chronic nasal discharge during their childhood age. Two cases had history of operation of deviated nasal septum while none of them had syphilis, tuberculosis or leprosy and any history of accident.

Socio–economic status

Out of the 25 cases, 20 came from the low socio-economic status group living on poor diet and in unhygienic conditions. Rest of the 5 cases belonged to middle class society while none belonged to the rich class.

Table No. – VIII

Socio-economic status	No. of patients	Percentage
Low	20	80%
Middle	5	20%
High	-	-
Total	25	100%

Unilateral Atrophic Rhinitis

Only one patient had unilateral atrophic rhinitis due to a markedly deviated nasal septum, rest of cases had no septal deviation in this series.

Bacteriological Profile

We had studied 25 cases of atrophic rhinitis and nasal swab were taken before medication. Table No.IX shows incidence of different types of Bacteria in the atrophic rhinitis.

Table No. – IX

Type of Bacteria	No. of patients	Percentage
Pseudomonas aeruginosa	20	80%
E. Coli	1	4%
Proteus mirabilus	1	4%
Staphylococcus aureus	2	8%
Sterile	1	4%
Total	25	100%

Out of the 25 cases one Nasal Swab was sterile (4%) because patients had taken treatment from nearby doctors and than came to us for management. The commonest bacteria isolated in this study was Pseudomonas aeruginosa in 80% cases. Staphylococcus aureus was isolated in 8% cases. Proteus mirabilus was isolated in 4% cases and E. Coli was isolated in 4% cases.

Radiological Examination of Paranasal sinuses

Only one patient had normal maxillary antrum and rest 24 patients had small maxillary antrum with thick bony walls. Frontal sinus was absent in 12 cases, small in 12 cases and normal in 1 case. Table No.X shows following data.

Table No.-X

Sinus development		No. of pat-ients	Severity of disease						
			Age	Crust in nose	Atrophy of turbinates	Anosmia	Hyposmia	Ozaena	%
Frontal Sinus	Absent	12	18-70	Severe	Present	Present		Present	48%
	Small	12	11-70	Moderate	Present	Present		Present	48%
	Normal	1	25	Mild	Present	Present	Present	Present	4%
Maxillary Sinus	Normal	1	11	Mild	Present	Present		Present	4%
	Small	24	18-70	Moderate to severe	Present	Present		Present	96%

Maxilary antrum was hazy in 8 cases i.e. maxillary sinusitis in 8 cases and in rest of the cases maxillary antrum was not hazy. Frontal sinus was hazy in 4 cases and in remaining patients it was clear. Table No.XI shows the following feature.

Table No. – XI

Type of Sinusitis	No. of Patients	Percentage
Maxillary Sinusitis	8	32%
Frontal Sinusitis	4	16%
Bone erosion or destruction	12	48%

Erosion of bony parts of septum and bony turbinates was seen in 12 cases (48%).

Antrum Punctures

We had done antral wash in 24 patients. In 12 patients the return was clear fluid and in 4 patients the returning fluid contained mucus. The remaining 8 cases had pus on wash out.

This is shown in Table No.XII.

Table No. – XII

Type of fluid on antrum wash	No. of Patients	Percentage
Clear	12	50%
Mucus	4	16.7%
Pus	8	33.3%
Total	**24**	**100%**

PREVALENCE OF PRIMARY ATROPHIC RHINITIS AND SECONDARY ATROPHIC RHINITIS

One patient had deviated nasal septum (severe) to one side and two patients had history of operation for deviated nasal septum. Rest of the patients had no signs of septal deviation. This is shown in Table No.XIII.

Table No. – XIII

Type of Atrophic Rhinitis	No. of Patients	Percentage
Primary Atrophic Rhinitis	22	88%
Secondary Atrophic Rhinitis	3	12%

Most of the patients had no deviation of nasal septum, no history of septal operation, diseases like leprosy, tuberculosis, syphilis and any accidents. Therefore most common type of atrophic rhinitis was Primary Atrophic rhinitis in (88%) of the cases of this series and 12% patients had secondary atrophic rhinitis.

Histopathology

Variation in Epithelium

Table – XIV

Change in Epithelium

Change in Epithelium	No of patient	Percentage
Normal Respiratory epithelium	1	4%
Squamous	21	84%
Transitional	3	12%
Total	**25**	**100%**

Epithelium was normal pseudostrafified tall columnar with reduced number cilia and also with less goblet cells in 1(4%) case. There was squamous metaplasia in 21 (84%) cases. Transitional epithelium was seen in 3 (12%) cases.

Condition of Cilia

Table No.XV

Showing Changes in Cilia

Change in Cilia	No. of Patient	Percentage
Normal	–	–
Reduced In Number	1	4%
Absent	24	96%
Total	**25**	**100%**

There was reduced number of cilia seen in 1 (4%) cases. Cilia was absent in 24 (96%) cases of this series.

Basement membrane :-

Table No. 16 showing Change in the basement membrane.

Table No.XVI

Change in basement membrane	**No. of cases**	**Percentage**
Normal	1	4%
Thin	19	76%
Thickened	5	20%
Total	**25**	**100%**

Basement membrane was normal in 1 (4%) cases on routine H and E staining section. A hyaline thick pinkish band like structure was observed in 5 (20%) cases. A thin band like basement membrane was seen in 19 (76%) cases of this study .

LAMINA PROPRIA

A) **Cellular Infiltrate :**

Table No.XVII showing Prominent Cell Infiltrate.

Table No.XVII

Prominent Cell	No. of Patient	Percentage
Lymphocytes	23	92%
Plasma cells	2	8%
Eosinophils	–	–
Neutrophils	–	–
Total	**25**	**100%**

Cell infiltration was seen mostly in subepithelial zone or widely distributed deep into the lamina propria.

The lymphocytes were most prominent cell in 23 (92%) cases. Plasma cells was most prominent cell seen in 2 (8%) Cases but also seen in 10 (40%) Cases. Eosinophils were not prominent in anyone case but seen in 3 (12%) cases.

B) VASCULAR CHANGES

Table No. –XVIII

Changes in Blood Vessels

Vascular Changes	No. of Patients	Percentage
Normal	2	8%
Dialated	13	52%
Endarteritis	8	32%
Periarteritis	2	8%
Total	**25**	**100%**

Blood vessels were normal in 2 (8%) cases. Dialation of vessels were noted in 13 (52%) cases. Endarteritis of terminal arterioles were seen in 8 (32%) cases and periarteritis was seen in 2 (8%) cases.

C) **GLANDULAR CHANGES**

- TABLE NO – 19 SHOWING CHANGES IN THE GLANDS

Table No. XIX

Change in glands	**No. of Patients**	**Percentage**
Normal	2	8%
Atrophy & reduced in Number	10	40%
Absent	13	52%
Total	**25**	**100%**

Glands were normal proportion in 2 (8%) cases. Atrophy and reduced in number was observed in 10 (40%) cases. Glands were absent in 13 (52%) cases.

D) **FIBROSIS :-**

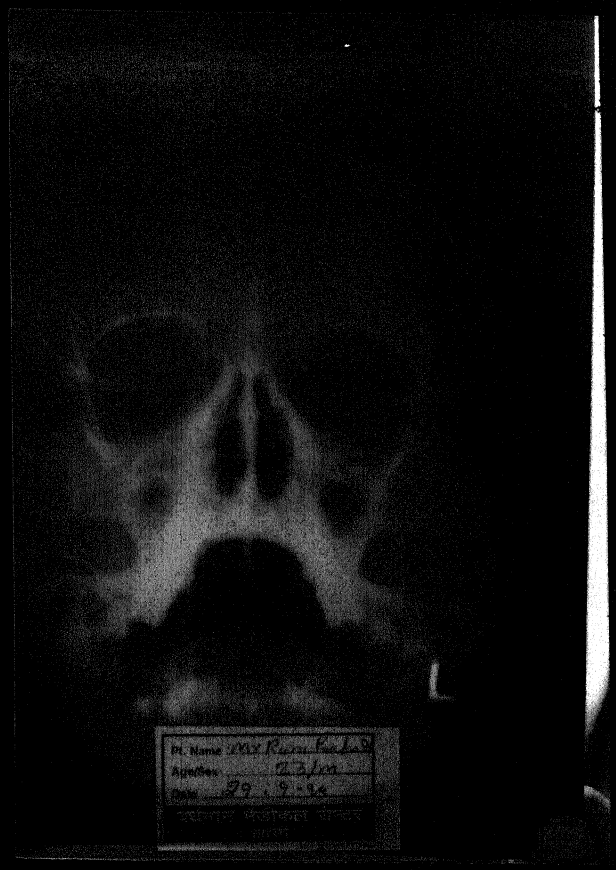
Table No.XX

Showing Degree Of Fibrosis

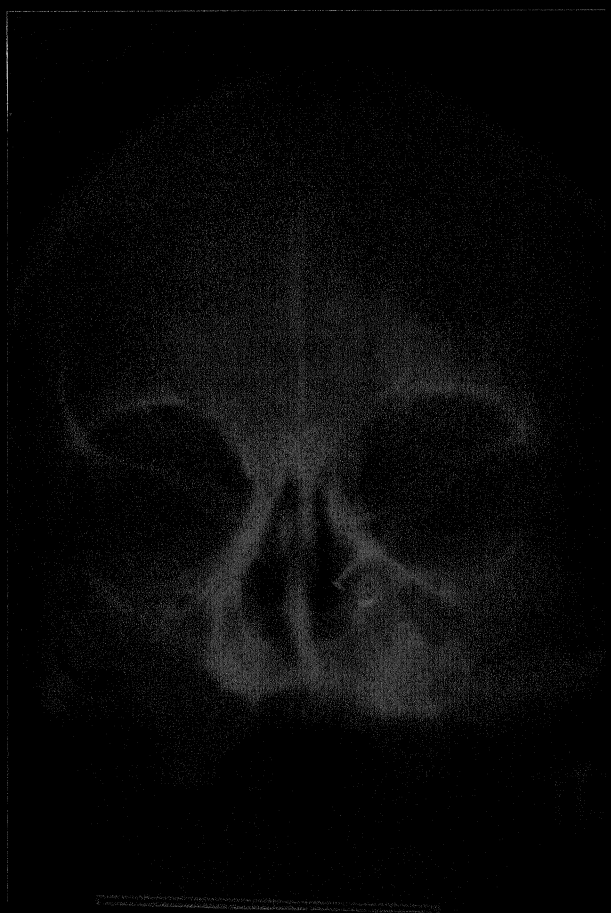
Degree of Fibrosis	No. of Patients	Percentage
Mild	12	48%
Moderate	10	40%
Severe	3	12%
Total	**25**	**100%**

Different degree of fibrosis was present in this study. It was graded as mild, moderate and severe.

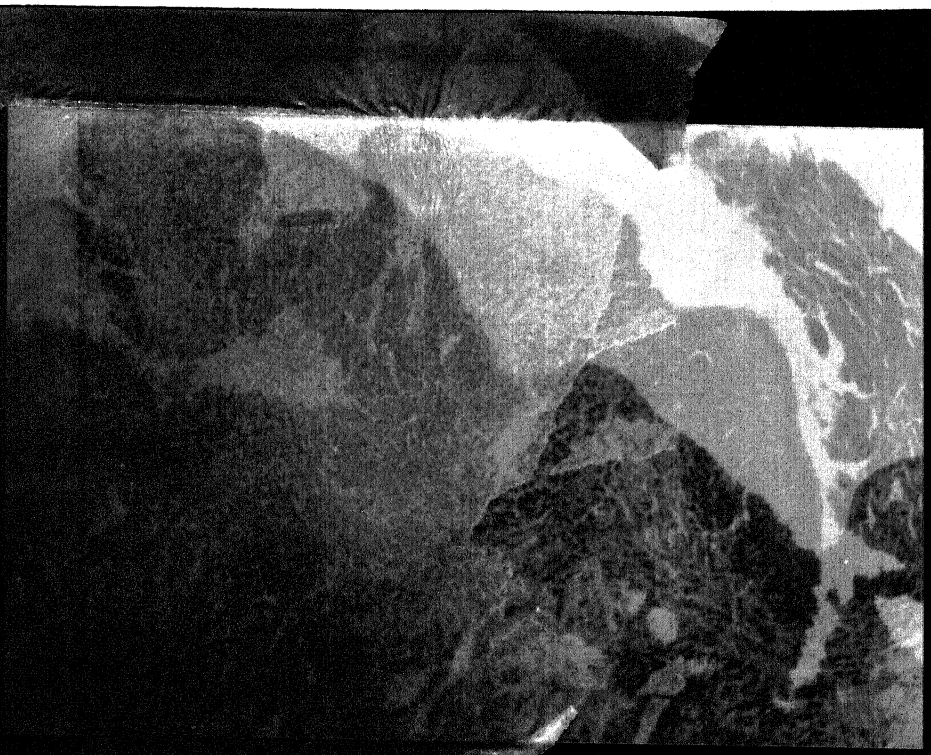
In 12 (48%) cases it was mild degree, in 10 (40%) cases moderate and 3 (12%) cases had severe degree of fibrosis.



X-RAY PNS OCCIPITOMENTAL VIEW SHOWING SMALL MAXILLARY ANTRA WITH ABSENT FRONTAL SINUS



X-RAY PNS OCCIPITOMENTAL VIEW SHOWING SMALL MAXILLARY ANTRA WITH ABSENT FRONTAL SINUS



MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
SQUAMOUS METAPLASIA (H&E STAIN. 7 x 10 X)

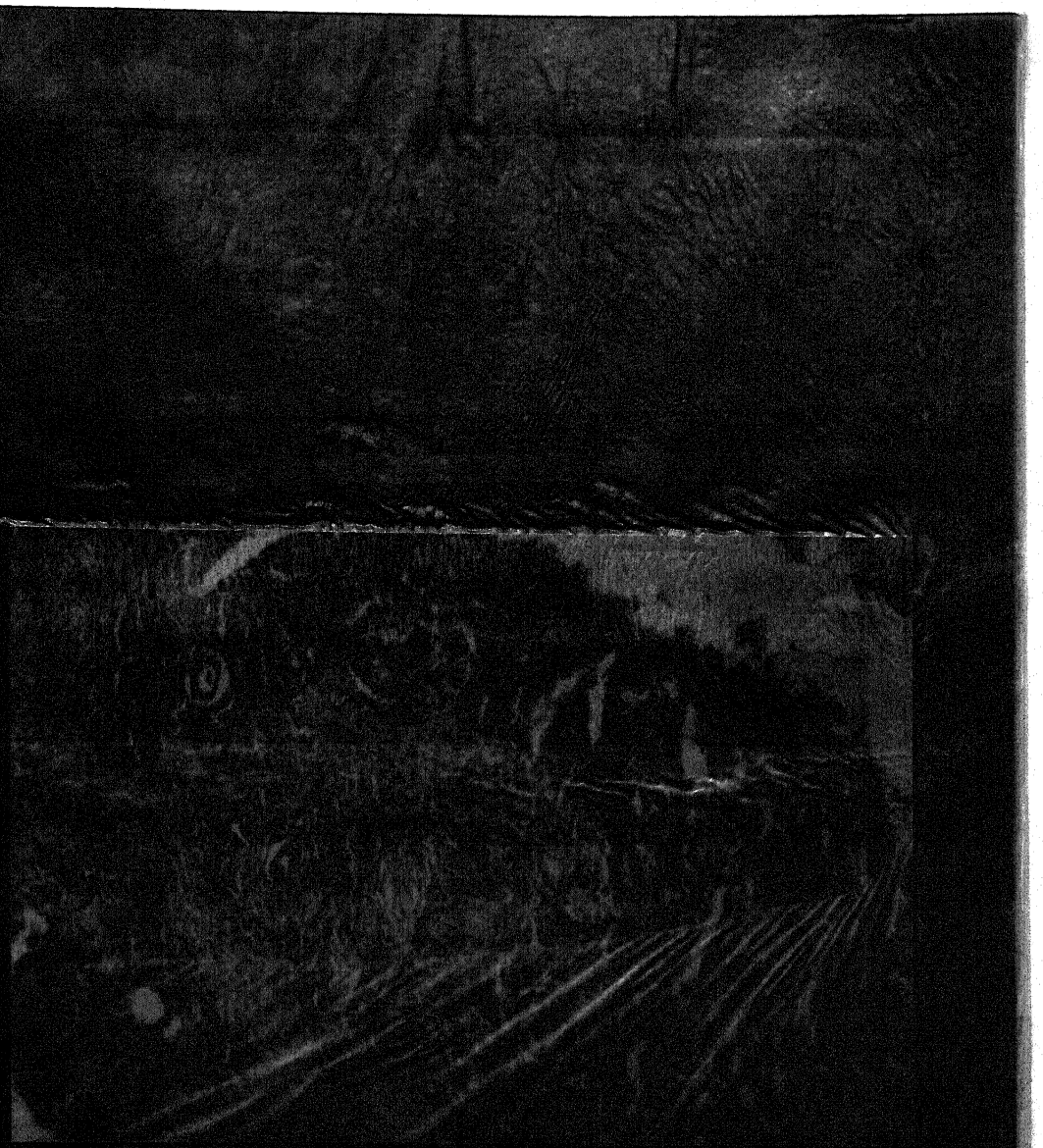
MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
THICK BASEMENT MEMBRANE (H&E STAIN. 7 x 10 X)

MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
THIN BASEMENT MEMBRANE (H&E STAIN. 7 x 10 X)

MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
DIALATED VESSELS (H&E STAIN. 7 x 10 X)

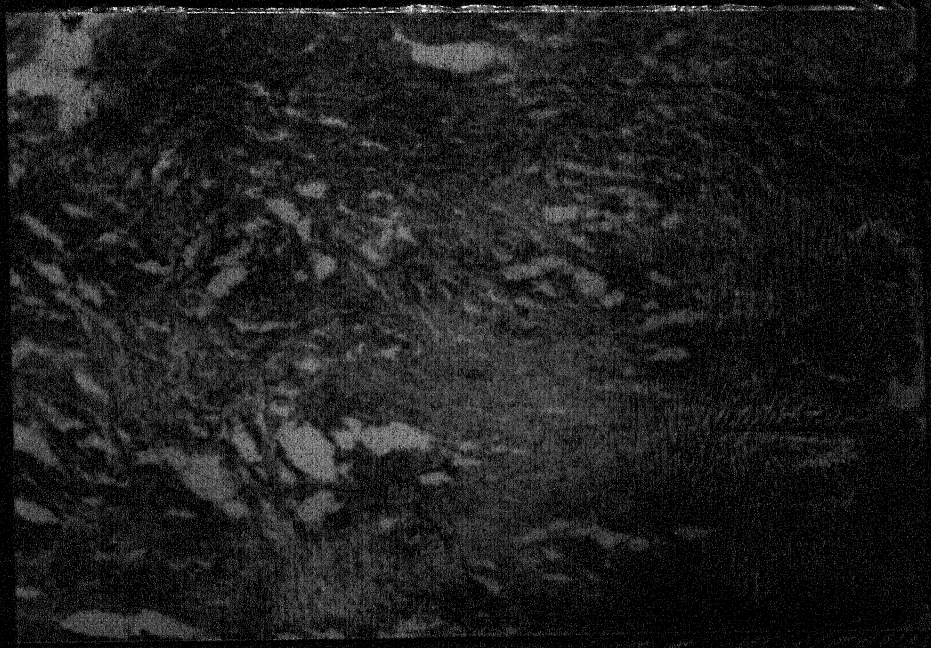
MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
ENDARTERITIS (H&E STAIN. 7 x 10 X)

MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
LYMPHOCYTE INFILTRATION (H&E STAIN. 7 x 10 X)



MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
ATROPHY OF GLANDS (H&E STAIN. 7 x 10 X)

MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
ABSENCE OF GLANDS (H&E STAIN. 7 x 10 X)



MICROSCOPIC PHOTOGRAPH
OF ATROPHIC RHINITIS SHOWING
FIBROSIS (H&E STAIN. 7 x 10 X)

DISCUSSION

DISCUSSION

The present study was done on 25 selected cases of atrophic rhinitis. Only those cases were included in whom the symptoms were attributable to the atrophic rhinitis. Special attention was paid to find out the cause of atrophic rhinitis, whether it is primary or secondary type. Attempts were made to find out where ever possible, the factor responsible for causation of atrophic rhinitis. The diagnosis of atrophic rhinitis was made on the basis of clinical and histopathological examination.

INCIDENCE

Atrophic Rhinitis is one of the disappearing disease in the western countries. Western authors attribute this change to their improvement in living standards. Girgis (1966) recorded 1% incidence of atrophic rhinitis among OPD cases in United Arab Republic.

In our country the disease is still prevalent. Jain (1966) reported a frequency of one case of atrophic rhinitis for an average of 1700 new out patients.

In the present study the incidence was 0.5% among the new out patients cases.

Age Incidence

Atrophic Rhinitis is described as a disease of young subjects. Most of the authors believe that the disease usually begins about the age of puberty. According to Thomson and Negus (1955) the disease begins mostly between the age of 7 to 12 years and in a few after 25 years. In this series age of the patient ranged from 11 to 68 years.

In our series 6 cases (24%) belonged to the 21 to 30 years and 6 cases (24%) belonged to 51 to 60 years of age, 20% cases belonged to 31 to 40 years. 4cases (16%) belonged to 11 to 20 years of age.

The youngest of the series was 11 years and the oldest was 68 years. Even in the age group above 20 years the onset of the disease could be definitely taken back to an early age.

Sex Incidence

James (1965) has stated that the disease is 5 times more common in females than in males. In the present study shows 18 females out of 25 cases (72%). The disease is 2.57 times more common in females in our series. It seen that less caloric diet, early marriage, poor hygiene and non availability of medical facilities are few causes why it is common in females.

Religion

In present study 88% of the cases (22) of Atrophic rhinitis were Hindus and 12% (3 cases) were Muslims and no case of Christian and other casts was seen. The Muslim population of this area of Bundelkhand constitutes 11.5% of entire population (1971 census). This incidence of Muslims patients in the present study could be explained on the basis of population of this community seeking advice of treatment in this hospital not only from Jhansi but also from nearby places.

Symptomatology

The positive history of crusting in the nose was present in 100% of cases. Rural were more affected than Urban. History of foetor was present in all cases (100%) and also Anosmia in 96% cases. Nasal blockage was present in 92%, Nasal discharge in 72% cases and headache was present in 76% of the cases. Dryness of pharynx or throat was present in 80% cases.

The alarming symptoms seeking attention of the patients i.e. bleeding per nose and myiasis of nose was present in 40% of cases. Epistaxis was mostly associated with long history of complaints and myiasis of nose were more common in older age group. Frequent attack of cold and sneezing were present only in 28% of the cases. The disease was bilateral in 96% of cases and unilateral in 4% of cases. Hoarseness of voice was not present in any case. Atrophy of pharyngeal mucosa was found in 20 cases (80%).

Why maggots infestation occur in old age group can be explained that most of older population is neglected in families and hygiene suffer again because they can not take care of their own cleaning.

Occupational Incidence

Girgis (1966) has stated that the disease is more common in farmers i.e. poor citizens. In our series majority of the patients were house wives 64%, followed by farmers 24%, students of 8% and Bidi maker 4%. They were mostly working in dry condition.

Seasonal factor

Although disease run for whole of year but complaint are more in two season in Bundelkhand, one after winter season and other after Rainy season and before winter set in.

Duration of Illness

In the present study the majority of the patients presented with long duration 56% (14 cases) while few cases had disease with short duration 12% (3 cases). So it can be concluded that atrophic rhinitis is a chronic disabling disease.

Predisposing Factors

Most of the cases of this series belonged to poor socio-economic group 80%, while few belonged to the middle class 20%. The fact that none belonged to the rich class is significant. Poor hygienic conditions, climate (dry) substandard nutrition are all may be predisposing factors but infections disease per se do not contribute much to the incidence.

One case had severe septal deviation. Which lead to the development of atrophic rhinitis to opposite side and two cases had history of septal surgery.

Relationship between the disease and familial tendency has been suggested by Turner (1960) and many other authors. We had in our series 13 (52%) cases having family members suffering from the Atrophic rhinitis. Hence it can be said that the factor of heredity does play some role in causing atrophic rhinitis.

It is surprising to find that despite Tuberculosis rampent in Bundelkhand region, neither there was any clinical evidence of Tuberculosis nor there was any past history or family history of Tuberculosis in any of these patients. This is definitely contradictory to the observations of Dan Makenzie (1916) and Negus (1956). In this series none of the patient had leprosy, syphilis and other chronic disease.

Antrum Puncture Wash

Antrum wash out in most of the cases were difficult. This is explained by the selerosis of the Bony Wall of the Maxillary antrum (Tapen and Apte 1969). We agree with their statement that when the antrum puncture proved difficult in these cases, the return came clear most of the times 50% (12 cases). Pus came in 8 cases i.e. 33.3% and mucus came in 16.7% (4 cases).

Prevalence of different Bacteria in Atrophic Rhinitis

In the past numerous organisms have been cited as the cause among which are Cocco-bacillus (Loewenberg, 1894), Bacillus mucosus (Abel, 1895), Cocco bacillus foetidus ozaena, diptheroid bacilli and Klebsiella ozaenae (Henriksen and Gundersen, 1959).

In the present study the commonest Bacteria was Pseudomonas aeruginosa 80% (20 cases) followed by staphylococcus aureus 8% (2 cases) and other bacteria were E. coli 4% (1 case) and Proteus mirabilus 4% (1 case). In one case (4%) the nasal swab was sterile. The infection is transmitted by contact or passively by flies. Bernet (1968) felt that the Bacteria had no role to play in the pathogenesis of atrophic rhinitis. It was difficult to conclude from the present study whether bacteria were causative agents of atrophic rhinitis or were secondary invaders. The etiological role of bacteria can only be confirmed or excluded by reproducible experimental studies in animals.

Prevalence of Primary and Secondary Atrophic Rhinitis

Atrophic Rhinitis has been divided into two groups on the basis of the causative factors (James, 1965). Primary atrophic rhinitis includes those cases where exact cause is obscure. It is quite possible to consider multiple factors to produce this conditions while cause of secondary atrophic rhinitis is known.

In the present study majority of the cases were primary atrophic rhinitis 88% (22 cases) and only 3 case had secondary atrophic rhinitis 12% out of which one had gross septal deviation and two had history of DNS operation.

In atrophic rhinitis the nose is affected bilaterally but in presence of gross septal deviation the condition may be absent in the narrow fossa (Thomas and Negus 1955). In present study one patient had gross deviation to left side and atrophic rhinitis on the right side.

Only 10 cases (40%) gave history of chronic nasal ailment prior to the onset of the disease. However only two had sinusitis, when examined first.

Radiological Findings

In the present study majority of the cases had small maxillary antra with thickened wall 96% (24 cases) and one had normal size maxillary antra 4%.

Peste (1949) demonstrated that large nasal fossae in atrophic rhinitis are at the expense of small under developed antra. The author conclude that the poor pneumatisation of antra is probably the decisive factor in the pathogenesis of the disease.

Frontal sinus was absent in 12 case (48%) and small in 12 cases (48%) while 1 case (4%) had normal sized frontal sinus. Frontal sinus was hazy in 16% cases (4 cases) and remaining were clear. Maxillary antra were hazy in 32% cases (8 cases) and rest of the cases had clear maxillary antra.

Erosion of Bony part of septum, turbinates was seen in 48% (12 cases). Wachsberger (1934) stressed the importance of abnormal width of the nasal cavities.

We also found widened nasal cavity in 96% (24 cases) of this series. In one case we could not find wide nasal cavity.

Our findings are accordance with Peste observation i.e. small maxillary antra with thickened wall found nearly in all cases.

Therefore it can be concluded that poor pneumatisation of maxillary antrum and wide nasal cavity produces definite predisposition to the development of atrophic rhinitis. Nasal cavity is normally kept wet by mucous production which in normal humidity is approximately 1250ml Bilateral side. Now demand increases when humidity is less (dry season) probably small maxillary antrum produces small quantity of mucous. That explain the radiological finding with dryness of cavity.

It can be explained that there could be some hereditary cause where less amount of mucqus gland and mucous production is relation to small sinuses makes patient vulnerable to dry incoming air. Specially in dry season leads to atrophic rhinitis.

HISTOPATHOLOGY

1. *Epithelium*

variation in covering epithelium was seen in atrophic rhinitis. Its was normal with reduced number of cilia also with less goblets cells in 1 case (4%), Transitional metaplasia observed in 3 (12%) cases while squamous metaplasia were seen in 21 (84%) cases. According to Eggston and Wolf (1947), sqamous metaplasia is commonly occurred in atrophic rhinitis. The finding of present study agreed with eggston and Wolf in this regard.

2. *Basement Membrane*

Hyaline thickning of basement membrane was seen in 5 (20%) cases. Basement membrane was normal in 1 (4%) case. Thin basement membrane was seen in 19 (76%) cases in this series. Taylor and Young (1961) have found the basement membrane to be thin. Others like Shambaugh (1931), Hollender (1944) have noted thickening of this membrane due to more collagen. According to this study the finding of Taylor and Young is more true than others.

3. *Lamina Propria*

(a) Cellular Infiltrate

Lymphocytes are predominantly seen in atrophic rhinitis (92%) plasma cells is predominant cells in (8%) while Easionphils and plasma cells were seen in some cases. Therefore, it can be concluded that atrophic rhinitis is a chronic inflammatory disease of nose.

(b) Changes in the vessels

The vessels were normal in 2 (8%) cases, endarteritis of terminal arterioles was seen in 8 (32%) cases while periarteritis was seen in 2 (8%) cases. According to Ruskin, 1942, Halopainen (1967) it is due to chronic inflammation of vessels. Dialated vessels were seen in 13 (52%) cases of this series. Taylor and Young (1961) also found dialated capillaries in his study. So we confirmed the statement of Taylor and Young, Ruskin and Halopainen changes in vessels are due to chronic inflammatory process.

(c) Glandular Changes

there is a decrease in the number and size of compound alveolar glands (Taylor and Young, 1961). In the present study we found glands normal in size and shape in 2 (8%) cases. Atrophy of glands and reduction in number found in 10 (40%) cases. Glands were absent in 13 (52%) cases. Dryness of nasal mucosa was found nearly all cases due to absence or less number of glands.

(d) Fibrosis

Fibrosis of lamina propria was seen in almost all cases. Severe degree of fibrosis were noted in 3 (12%) cases. Moderate degree of fibrosis was seen in 10 (40%) cases and mild degree of fibrosis was seen in 12 (48%) cases. Shambaugh (1931), Hollender (1944) have all reported variable degree of fibrosis in their study. The finding of present study concur accordingly.

SUMMARY AND CONCLUSION

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The present study comprised of 25 cases of atrophic rhinitis in Bundelkhand region who attended the E.N.T. O.P.D. of M.L.B. Medical College, Hospital Jhansi, U.P. during the period of January 1998 to January 2000.

The present study revealed the following facts :-

1. The overall incidence of atrophic rhinitis was 0.5% in E.N.T., O.P.D. during the study period.

2. The incidence of atrophic rhinitis was found more in young adolescents. The youngest of the series was 11 years old and oldest was 68 years.

3. Females were affected more (72%) as compared to males (28%).

4. Majority of the cases belongs to Housewives (64%), Farmer (24%) and rest of cases were from students (8%), Bidi maker (4%).

5. Atrophic rhinitis was more common in Hindus (88%) and Muslims (12%).

6. Climate, poor hygienic and socio-economic conditions (80%), positive family history are all factors to be reckoned with this disease. Syphilis, leprosy and Tuberculosis were not a major factor in the incidence.

7. The presenting symptoms of atrophic rhinitis were crusting (100%), Foetor (100%) Anosmia (96%), Nasal blockage (92%), Dryness of throat (80%) and headache (76%).

8. Majority of the patients (56%) presented with disease since long duration.

9. On examination the most common findings were crust in the nose (100%), Foetor (100%), Dryness of nasal mucosa (100%), Widened nasal cavity (96%), Atrophy of turbinates (100%), Pale colour of nasal mucosa (96%) and atrophy of pharyngeal mucosa in (80%) cases.

10. Majority of the cases had bilateral atrophic rhinitis (96%) than the unilatreal (4%).

11. Prevalence of primary atrophic rhinitis was 88% and secondary atrophic rhinitis was 12%.

12. The commonest bacteria was isolated Pseudomonas aeruginosa (80%), followed by staphylococcus aureus (8%), rest were proteus mirabilus (4%), E. Coli (4%) and Sterile (4%).

13. Frontal sinus was normal in 1 (4%) small in 12 (48%) and absent in 12 (48%).

14. Most of the cases had small underdeveloped maxillary antra in 24 (96%) with thick bony walls and normal in one (4%).

15. The maxillary antrum was hazy in 32% cases, and frontal sinus in 16% cases. In remaining cases sinuses were clear.

16. Squamous metaplasia was found in 84% (21) cases, while transitonal in 12% (3) cases.

17. Thin basement membrane was seen in 76% (19) cases and thick in 20% (5) cases.

18. Cell infiltration was seen in lamina propria. The lymphocytes were most predominent cells in 92% (23) cases followed by plasma cells in 8% (2 cases).

19. Dialated blood vessels were seen in 52% (13) cases followed by endarteritis in 32% (8) cases, Periarteritis was seen in 8% (2) cases.

20. Alveolar glands were absent in 52% (13) cases, reduced in size and number were seen in 40% (10) cases.

21. Fibrosis of lamina propria was mild degree in 48% (12) cases, moderate degree in 40% (10) cases and severe degree of fibrosis in 12% (3 cases).

The available literature regarding the clinical feature, bacteriological profile, aetiology and histopathology of atrophic rhinitis was reviewed and the findings of the present study were interpreted in the light of the same.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Abel, P. (1895). Die aetiologic der ozaeria. Zertschrift fur Hygiene and Infektionskrankheiten 21: 89-95.

2. Arnstein , A. R. (1972) : regional osteoporosis, Orthopaedic Clinics of North America, 3 : 585.

3. Boeck C, (1905) Fortgetzte untersuchungen uber das multiple benigne sarkoid. Archiv fur Dermatologie and Syphilis, 73, 301-332.

4. Ballenger, H.C., Ballenger, J.J., 1948. Disease of Ear, Nose and Throat, Ed. Philadelphia. Lea & Fegiber, Page 45-57.

5. Bailey, H & Love, M. (1959) : A short practice of Surgery, J & K Lewis & Co. Ltd., London. P-1191.

6. Bernat, Ivan (1965) : Ozaena, A manifestation of Iron Deficiency, pergamon Press, Oxford, First English edition, P-4.

7. Bernat, J. (1965) Ozaena, a Manifestation of Iron Deficiency. Oxford: Pergamon Press.

8. Berkstein A. The treatment of atrophic rhinitis with injectable silicone, J. Laryng ; 80 : 6. 634, 1966.

9. Bergan, J. J & Conn. J. (1968) : Sympathectomy for pain relief, Medical Clinic of North America, 52 : 147.

10. Bernat, 1. (1968): Ozaena, a manifestation of Iron defliciency. Oxford, pergamon press, 13-14.

11. Ballenger. J.J., 1969. Disease of Nose, throat and ear. Ed 11. Philadelphia. Lea & Febiger, Page 13-20.

12. Barbary, A.E.S., Yassin, A., Fauad, H. and Shennawy, M.E. (1970) Histopathological and histochemical studies on atrophic rhinitis. Journal of Laryngology and Otology, 84: 1103-1112.

13. Barton R.P.E., Davey. T.F., McDougall. A.C., Rees, R.J.W. and Weddell, A.G.M. (1973) Clinical and histological studies of the nose in early lepromatous leprosy. Paper 6/47. Tenth International Leprosy Congress, Bergen.

14. Barton R.P.E. (1974) Olfaction in leprosy. Journal of Laryngology and Otology. 88, 355-361.

15. Barton R.P.E. (1976) Clinical manifestation of leprous rihinits. Annals of Otology, St Louis, 85, 74-82.

16. Barton R.P.E. and Davey, T.F. (1976) Early leprosy of the nose and throat. Journal of Laryngology and Otology. 90, 953-961.

17. Barton R.P.E. (1979) Radiological changes in the paranasal sinuses in lepromatous leprosy. Journal of Laryngology and Otology. 93, 597-600.

18. Brain, D.J. and Rock, W.P. (1983) The influence of Nasal trauma during childhood on growth of the facial skeleton. Journal of Laryngology and otology, 97, 917-923.

19. Boyette D. Morton (1983) : Zinc requirements in trauma and inflammation, Laryngoscope, 92 : 648-649.

20. Barton R.P.E. (1985) Ear, nose and throat involvement in leprosy. In: Leprosy, edited by R.C. Hastings. Edinburgh: Churchill Livingstone. Pp. 243-252.

21. Cottle, M.H., Fischer, G.G., Gaynon, I.E. and Loring,R.M. (1958) The 'Maxilla-Premaxilla' approach to extensive nasal septal surgery. Archives of Otolaryngology, 68: 301.

22. Cottle, M.H. (1958) Nasal atrophy, atrophic rhinitis, ozaena: medical and surgical treatment. Journal of the International College of Surgeons, 29: 472-484.

23. Chugg, C.M. et al. Surgical treatment of atrophic rhinits with injectable Silicone, Arch. Otolaryng ; 80 : 106, 1964.

24. Curtis, G.T. (1964) sarcoidosis of nasal bones. British Journal of Radiology. 37, 68-70.

25. Chatterjee, P.K. Katua, C.R. Chartterjee., S.N. and Dasidar, N. (1977) Recurrent multiple rhinosporidiosis with osteolytic lesions in hand foot. Journal of Laryngology and Otology. 94, 737-749.

26. Condas, S & Holland C. (1977) : Zinc : Clinical consideration, Osteopathic Medicine (Aug.) – quoted by Boyette (vide supra).

27. Chatterji, P. (1980) Autogenous medullary (cancellous) bone graft in ozaena. Journal of Laryngology and Otlogy, 94: 737-749.

28. Churchill-davidson, H.C. (1984) : A practice of anaesthesia, Fifth edition, Lloyd-Luke (Medical Books) Ltd., London. P-918.

29. Doupe, J., Gullen, G. H & Chance, G.O. (1944) Posttraumatic pain and the causalgic syndrome, Journal of Neurology, Neurosurgery and Psychiatry, 7 : 33.

30. Desa, Sandra, 1968. Evaluation of antral lavage in maxillary sinusitis. IJLO 20; 3, 115.

31. Douek, E. (1974) The sense of smell and its Abnormalities. London, Churehill Livingstone.

32. Datti, P.V. (1974). Closure of nostril in atrophic rhinitis. Indian Journal of Otolaryngology 26(4): 178-182.

33. Davey, T.F. and Rees, R. J. W. (1974) The nasal discharge in leprosy: clinical and bacteriological aspects. Leprosy review, 45, 121-126.

34. Eisenstode L.W. Surgical treatment of artophic rhinits, Arch Otolaryng; 40: 451, 1944.

35. Eggston A.A.and Wolf, D. (1947): Histopathology of the Ear, Nose and Throat, Williams & Wilkins Company.

36. Frederich, J. and Braude, A. (1974) Anaerobic infection of paranasal sinuses. New England Journal of Medicine 290, 135-137.

37. Fanous, N. and Baxter J.D. (1978) Silastic implant in atrophic rihinits. Journal of Otolaryngology, 7: 541-544.

38. Fouad, H., Afifi, N., Fatt-Hi, A., El-Sheeny, N., Iskander, I, and Abou Sail., M.N. (1980) Altered cell mediated immunity in atrophic rhinits, Journal of Laryngology and Otology, 94, 507-514.

39. Girgis, I.H. (1966). Surgical treatment of ozaena by dermato fat graft. Journal of Laryngology and Otology 80: 615-627.

40. Gadre K.C. et al. Colsure of the nostrils (young's operation) in atrophic rhinitis. J. Laryng 85 : 71, 1971.

41. Gadre K.G., Bhargava, K.B., Pradhan, R.Y., Lodaya, J. D & Ingle, M.V. (1971) : Journal of laryngology & Otology, 85 : 711.

42. Gardner, E., Gray, D. J & O'Rohilly, (1975) : Anatomy, 4th edition, W. B. Saunders Co., Philadelphia, P-735.

43. Gordan, W.W., Chon, A.M., Greenberg, S.D. and Komorn R. M. (1976) Nasal sarcoidosis. Archives of Otolaryngology, 102, 11-14.

44. Ghosh, P., (1980) : combined approach palatopharyngoplasty (CAP). Journal of Laryngology & Otologoy, 94 : 1165-1178.

45. Gray's Anatomy (1980) : Edited by Williams, P. L & warwick, R., 36th edition, Churchill Livingston, London P-1142.

46. Gwaltney, J.M. and Hayden, F.G. (1982) The nose and infection. In : The Nose: Upper Airway Physiology and the Atmospheric Environment, edited by D.F. Proctor and I. Andersen. Amsterdam: Elsevier Biomedical Press. Pp. 399-422.

47. Ghosh, P. (1986) ; Vestibuloplasty, a new one-stage operation for atrophic rhinitis, in press, Journal of Laryngology & Otology.

48. Ghosh, P. (1987). Primary atrophic rhinitis (with a new hypothesis for its aetiopathogenesis). Indian Journal of Otolaryngology 39: 7-13.

49. Heniriksen, S.D., gundersen, W.B., (1959). The aetiology of ozaena. Acta Pathologica et mirobiologica scandinavica 47: 380-386.

50. Holopainen, E. (1967) : Acta Otolaryngologica. (Stock), Suppl. P-227.

51. Helal, B. (1969) Silicones in orthopedic surgery. Recent advances in Orthopedics. Graham A.A. ed. PP. 91-94.

52. Huizing, E.H. (1969). Some conclusions from our experience with the surgical treatment of ozaena. International rhinology, 7: 81-87.

53. Jakabfi, I. (1954) : The problem of Ozaena (in Hungarian) Otorhinological Congress. Budapest, (Quoted by Bernat, 1. 1965).

54. James, D.G. (1959) Dermatological aspects of sarcoidosis. Quarterly Journal of Medicine, 28, 109-124.

55. Jensen, C. and Sydow, C. Von (1987) Radiography and ultrasonography in paranasal sinusitis. Acta radiologica, 28, 31-34.

56. Kistner, F. B. and Robertson, T.D. (1938) Benign granuloma of the nose, Journal of the American Medical Association, 111, 2003-2005.

57. Kameswaran, S. Surgical treatment of atrophic rhinits with OSSAR, Indian J. Otolaryng, 19: 130, 1967.

58. Kozin, F., McCarty, D.J., Simons, J. & Geunant, H. (1976) : The reflex sympathetic dystrophy syndrome I, Clinical & histological studies. Evidence of bilaterality, response to corticesteroids and articular involvement. American Journal of Medicine, 60 : 321.

59. Kasarkis, E.J. & Schuna, A (1980) : Serum alkaline phosphatase after treatment of zinc deficiency in humans, American Journal of Clinical Nutrition, 33 : 2609-2612.

60. Loewenberg, B. (1894) Le microbe de I' ozene. Annales de I' Institut Pasteur, 8, 292-347.

61. Moller-Christensen, V., Bakke, S.N. and Melson, R.S. (1952)Changes in the anterior nasal spine and alveolar process of the maxillary bone in leprosy. International Journal of Leprosy, 20, 335-337.

62. Messerklinger, W. (1966) Uber die drainage der menschlichen nasennebenhohlen 1. Mitteilung. Monatsschrift fur Ohrenheilkunde und Laryngo-Rhinologie, 100, 56-68.

63. Munro Black, J.I. (1966) Sarcoidiosis of the nose. Journal of Laryngology and Otology, 80, 1065-1068.

64. Miles Foxen. E.H. Diseases of the ear, nose and throat, ed. Scott Brown, Ballantyne J and Groves J. 3rd edition volume 3 London, Butter worths, 1971 page 126:

65. Mcdougall, A. C. Rees, R J. W. Weddell, A.G.M. and Wadji Kanan, M. (1974) The histopathology of lepromatous leprosy in the nose. Journal of Pathology, 115, 215-226.

66. Neil, J.F. (1967) Unilateral atrophic rhinitis. Journal of Laryngology and Otology, 81: 551.

67. Neil Weir. (1987): Scott Brown's Otolaryngology, London, Butter worth, 115-141.

68. Proud, G.O. Acrylic resin implant for atrophic rhinits, Laryngoscope 57:256, 1947.

69. Prasad, A.S., Rabin, P., Abbassii, A. (1978) : Annals of Internal Medicine 89 : 483-487.

70. Prasad, S. Ananda (1979) : Clinical Biochemical & Pharmacological Role of Zinc, Annual Review. Pharmacology & Toxicology, 20 : 393-426.

71. Pedersen, H. and Mygind N. (1982) Rhinitis, sinusitis and otitis media in Kartagener's syndrome (primary ciliary dyskinesia). Clinical Otolaryngology, 7, 373-380.

72. Petruson, B., Hansson, H.A. and KARLSSON, G. (1984) Structural and functional aspects of cells in the nasal mucociliary system. Archives of Otolaryngology. 110. 576-581.

73. Qizibash, A.A.H. and DAIF, M. (1992) atrophic rhinitis revisited. Pakistan Journal of Otolaryngology. 8. 197-199.

74. Ruskin, S.L. (1942) : Rationale of oestrogen therapy in primary atrophic rhinitis (ozaena). Arch. Otolaryng. 36 : 632.

75. Rethi, A. (1948) Operative treatment of ozaena Journal of Laryngology and Otology, 62: 139-146.

76. Roth, P. & Kirschgesner, M. (1974) : Zinc metalloenzymes in response to depletion & repletion of zinc, in Hoekstra, J.W. (Ed.), International symposium on trace element metabolism in Animals, Baltimore, University Park pess, P-509.

77. Rook, A. & Wilkinson, D.S. (1979) : Text Book of Dermatology Vol-2, P-20 15-2016, Blackwell Scientific Publication, Oxford.

78. Sack, N. (1927) : Zur frage der Aetiologic der stinknase, Z. HalsNas-u. Ohrenheilk, 10 : 181.

79. Sirala U. A. Operative method in the treatment of Ozaena, Arch, Otolaryng 72 : 2, 188. 1960.

80. Sharma, A.N. and Sardana, D.S. (1966) Stellate ganglion block in atrophic rhinitis. Journal of Laryngology and Otology 80, 184-186.

81. Sardana D S. et al : Acrylic resin implants in atrophic rhinitis Indian. J. Otolaryng 19:267, 1967.

82. Smith, A.B., (1968). In Logan Stewart's Diseases of the Nose, Throat and Ear, 7th ed., P. 44. Ed, by J.P. Stewart, Wright, Bristol.

83. Ssali, C.L. (1973). atrophic rhinitis: A new curative surgical treatment (middle turbinectomy). Journal of Laryngology and Otology 87: 397-403.

84. Shah, J.T., Karnik, P.P., Chitale, A.R. and Nadkarni, M.S. (1974) Partial or total closure of nostrils in atrophic rhinitis. Archives of Otolaryngology, 100, 196-198.

85. Sinha, S.N., Sardana, D.S. and Rajvanshi, V.S. (1977) Nine year review of 273 cases of atrophic rhinitis and its management. Journal of Laryngology and Otology, 91: 591-600.

86. Studdy, P., Bird, R., James D.G. and Sherlock, S. (1978) Serum angiotension-converting enzyme (SACE) in sarcoidosis and other granulomatous disorders. Lancet, ii, 1331-1334.

87. Schonsted-Madsen, U. Stoksted, P., Chirstensen, P.H. and Koch-henriksen, N. (1986) Chronic headache related to nasal obstruction. Journal of Larynology and otology, 100, 165-170.

88. Tos, M. (1892) Goblet cell and glands in the nose and paranasal sinuses. In: The Nose, edited by D. Proctor and I. Andersen. Amsterdam Elsevier: Biomedical Press, pp. 99-144.

89. Taylor, M and Young, A. (1961) Histopathological and histochemical studies on atrophic rhinitis. Journal of Laryngology and Otology, 75: 574-589.

90. Taylor, M. And Young, A (1961) Studies on atrophic rihinits. Journal of Laryngology and Otology, 75, 574-590.

91. Vogel, K. (1929) : Histopathologische Bofunde an Ganglion spenopalatinum mit besonderer Berucksichtgung der atrophischen Rhinitiden. Z. Hels-Nas., u. Ohrenheilk, 22 : 507.

92. Weiss, J.A. (1960) Sarcoidosis in otolaryngology. Laryngoscope, 70, 1351-1390.

93. Wilson, T.G. (1964) Journal of Laryngology and Otology, 78, 953.

94. Young, A. (1967) Closure of the nostrils in atrophic rhinitis. Journal of Laryngology and Otology, 81, 515-524.

95. Young, A. (1971) Closure of the nostril in atrophic rhinitis. Journal of Laryngology and Otology, 85: 715-718.

96. Zinreich, J. (1993) Imaging of inflammatory sinus disease. Otolaryngologic Clinics of North America, 26, 535-547.